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# Use of Cardiac Allografts With Mild and Moderate Left Ventricular Hypertrophy Can Be Safely Used in Heart Transplantation to Expand the Donor Pool

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**Objectives** The purpose of this study was to evaluate outcomes of heart transplantation (HTx) and changes in left ventricular wall thickness (LVWT) post-HTx using donors with left ventricular hypertrophy (LVH).

**Background** Limited data are available on use of donor hearts with LVH in HTx.

**Methods** We reviewed 427 patients who underwent HTx: 62 received hearts with LVH (interventricular septum [IVS] or posterior wall [PW] thickness ≥1.2 cm) by echocardiography, and 365 received hearts without LVH. The median follow-up was 3.8 years (range 0 to 16.2 years).

**Results** Recipient age was 56 ± 11 years and donor age was 30 ± 12 years. Baseline recipient characteristics were similar in both groups. Donors with LVH were older (35 ± 12 years vs. 29 ± 12 years, $p = 0.001$) and had higher rates of intracranial hemorrhage (38% vs. 15%, $p = 0.001$). The LVWT was increased in the LVH group compared with LVWT in the non-LVH group (IVS: 1.28 ± 0.18 cm vs. 0.85 ± 0.19 cm, PW: 1.27 ± 0.19 cm vs. 0.85 ± 0.20 cm, $p = 0.0001$ for both groups). Mild LVH (1.2 to 1.3 cm) was found in 42%, moderate (>1.3 to 1.7 cm) in 53%, and severe (>1.7 cm) in 5% of donors with LVH. Left ventricular wall thickness regression occurred in both IVS and PW (1.28 ± 0.18 cm vs. 1.10 ± 0.13 cm vs. 1.13 ± 0.14 cm, and 1.27 ± 0.19 cm vs. 1.11 ± 0.11 cm vs. 1.13 ± 0.14 cm, at baseline, 1 year, and 5 years, respectively; $p < 0.001$ for change from baseline to 1 and 5 years for both locations). Patients with or without donor LVH had similar 1-year (3.5% vs. 9.5%, $p = 0.2$) and 5-year survival rates (84 ± 5.9% vs. 70 ± 2.7%, $p = 0.07$).

**Conclusions** Short- and long-term survival rates and rates of LVH at follow-up were similar in both groups, suggesting that donor hearts with mild and moderate LVH can be safely used in HTx. (J Am Coll Cardiol 2008;51:1214–20)


Heart transplantation (HTx) provides definitive therapy for patients with end-stage congestive heart failure. Although improved preservation techniques and post-HTx immunosuppression have significantly improved outcomes, the number of patients on waiting lists has progressively increased over the last decade (1–3). Although initially strict donor and recipient selection criteria were established, liberalization of donor selection criteria and ways of expanding the donor pool have been suggested (4–7). Use of donor hearts with left ventricular hypertrophy (LVH) has varied among transplant centers, and there is little information concerning transplantation of donor hearts with LVH.

Until recently, only 2 small studies have been published and found that the presence of LVH in the donor heart was associated with early graft dysfunction and lower survival (8,9). Moreover, there are no data regarding the changes in measured wall thickness after HTx in this patient population. Given that use of donor hearts with LVH may permit an expansion of the donor pool, we aimed to review our experience with HTx using donors with LVH and to evaluate the changes in LVH over time.

## Methods

**Patients.** We retrospectively reviewed 427 consecutive HTx donors and recipients between 1989 and 2004 at Cedars-Sinai Medical Center. Recipients younger than 16 years of age and those with combined heart-lung transplan-

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tation were excluded. The donor and recipient evaluation and rejection surveillance have been described elsewhere (10). The post-HTx regimen included induction therapy consisting of OKT3 or Thymoglobulin. Maintenance immunosuppressive therapy included cyclosporine or tacrolimus, azathioprine or mycophenolate mofetil, and prednisone.

Donor hearts were preserved using a cold infusion of University of Wisconsin solution or Stanford solution prior to 2000. HTx was performed using the total orthotopic (bicaval) technique in 85% of recipients.

**Definition and grading of LVH.** Left ventricular hypertrophy was quantitatively assessed by echocardiography using wall-thickness measurements according to American Society of Echocardiography recommendations (11). Left ventricular hypertrophy was defined as interventricular septum (IVS) and/or posterior wall (PW) thickness ≥1.2 cm. Mild LVH was defined as wall thickness of 1.2 to 1.3 cm, moderate LVH as 1.4 to 1.7 cm, and severe LVH as >1.7 cm. Follow-up echocardiograms at 1 and 5 years post-HTx were available in 66% and 49% of patients, respectively. The electrocardiogram (ECG) definition of LVH was based on standard voltage criteria: SV1 + RV5 or RV6 >35 mm.

**Statistical analysis.** Results for continuous variables are presented as mean ± SD and for categorical variables as frequency (percentage). The comparison of continuous variables between groups was made using *t* test or the Wilcoxon rank sum test as appropriate. Categorical variables were compared using chi-square or Fisher exact tests. Within-group change in numerical variables across 2 time points was assessed by the Wilcoxon signed rank test. Within-group change in dichotomous variables across 2 time points was assessed by the McNemar test for related proportions. Survival estimates were generated by the Kaplan-Meier method. The log-rank and Wilcoxon tests were used to compare survival across groups. Multivariable stepwise Cox proportional hazards models were employed to assess variables associated with the risk of death. All statistical tests were two-sided, and a significance level of 0.05 was used throughout. Statistical analyses were performed using the SAS system version 9.1 (SAS Institute Inc., Cary, North Carolina).

## Results

**Donor and patient characteristics.** Sixty-two patients received a donor heart with LVH, and 365 received a heart without LVH. Median follow-up was 3.8 years (range 0 to 16.2 years).

Donors with LVH were significantly younger ($p = 0.0003$) and had a higher prevalence of intracranial bleeding ($p = 0.02$) (Table 1). No significant differences were observed in terms of other baseline characteristics. Twenty-nine donors (48%) had history of hypertension (HTN). Among donors with LVH, 42% of patients had mild, 53% had moderate, and 5% had severe LVH as determined by echocardiography. Twenty-five (40%) donors also had evidence of LVH by as determined by ECG.

Baseline pre-HTx recipient characteristics were similar in the 2 groups and are presented in Table 1. The post-HTx recipient characteristics are presented in Table 2. A long (>240 min) ischemic time was found only among patients who received allografts without LVH (6%). A larger number of patients with donor LVH were treated with tacrolimus ($p = 0.004$). No significant differences were found in terms of length of hospitalization, acute cellular rejection ≥3A, and cytomegalovirus infection rates.

**Abbreviations and Acronyms**

**ACEI** = angiotensin-converting enzyme inhibitor
**ARB** = angiotensin receptor blocker
**HTN** = hypertension
**HTx** = heart transplantation
**IVS** = interventricular septum
**LV** = left ventricular
**LVH** = left ventricular hypertrophy
**LVWT** = left ventricular wall thickness
**PW** = posterior wall

**Survival analysis.** No significant differences in 30-day and 1-year mortality were found between recipients of donor heart with LVH compared with those without LVH (1.6% vs. 3.3%, $p = 0.2$, and 3.5% vs. 9.5%, $p = 0.7$, respectively). The overall survival of the 2 groups is shown in Figure 1 and reveals no significant difference ($p = 0.07$) (Fig. 1A). Multivariable stepwise Cox proportional hazards analysis found evidence that donor LVH was associated with a reduced death hazard rate. The independent predictors of mortality are shown in Table 3.

Prior studies have demonstrated lower survival in recipients of older donor hearts (12). We divided both groups, with and without donor LVH, by donor age <45 years (younger) and ≥45 years (older). Survival analysis revealed a trend ($p = 0.08$) (Fig. 1B) indicating a possible difference among recipients of younger donors with or without donor LVH and older donors with or without LVH.

A separate analysis comparing survival among patients who received hearts with mild LVH or moderate or severe LVH did not reveal significant differences ($p = 0.82$). No significant difference in survival between recipients with donor LVH as determined by both ECG and echocardiography and those with donor LVH determined only by ECG evidence was observed ($p = 0.58$) (Fig. 2A). For about the first 2.5 years post-transplant, almost identical survival was observed among recipients of donor LVH with and without donor history of HTN; however, those with donor history of HTN have a trend for worse survival thereafter ($p = 0.05$) (Fig. 2B).

One hundred sixty-two patients died over the follow-up period: 13 patients (1 within 30 days) in the LVH group and 149 (12 within 30 days) of those in the group without LVH. The causes of late deaths were right ventricular failure (3%), rejection (10%), cardiac (36%), and noncardiac (51%). Compared with recipients of allografts without

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**Table 1 Baseline Characteristics of Donors and Recipients**

| | Donors With LVH (n = 62) | Donors Without LVH (n = 365) | p Value* |
|---|---|---|---|
| Donor characteristics | | | |
| Age (yrs) | 35.5 ± 12.0 | 29.5 ± 11.9 | 0.0003 |
| Male gender | 38 (61%) | 261 (72%) | 0.1 |
| BSA ($m^2$) | 1.95 ± 0.22 | 1.90 ± 0.25 | 0.2 |
| Caucasian | 32 (52%) | 201 (55%) | 0.7 |
| Intracranial bleeding | 24 (38%) | 56 (15%) | 0.02 |
| Cerebrovascular accident | 10 (16%) | 46 (13%) | 0.4 |
| Inotropic support | 56 (90%) | 338 (93%) | 0.6 |
| Thyroxine treatment | 18/61 (30%) | 115/336 (34%) | 0.6 |
| IVS thickness (cm) | 1.28 ± 0.18 | 0.85 ± 0.19 | 0.0001 |
| PW thickness (cm) | 1.27 ± 0.19 | 0.85 ± 0.20 | 0.0001 |
| Mild LVH (WT 1.2 to 1.3 cm) | 26 (42%) | | |
| Moderate LVH (WT >1.3 to 1.7 cm) | 33 (53%) | | |
| Severe LVH (WT >1.7 cm) | 3 (5%) | | |
| Recipient characteristics | | | |
| Age (yrs) | 56.9 ± 11.5 | 56.2 ± 11.4 | 0.7 |
| Male gender | 47 (75%) | 299 (82%) | 0.3 |
| Caucasian | 46 (74%) | 271 (74%) | >0.9 |
| Diabetes mellitus | 22 (36%) | 84 (23%) | 0.04 |
| Hypertension | 22 (36%) | 106 (29%) | 0.3 |
| Creatinine (mg/dl) | 1.52 ± 0.84 | 1.39 ± 0.77 | 0.1 |
| Ischemic cardiomyopathy | 32 (52%) | 190 (52%) | >0.9 |
| NYHA functional class III to IV | 59 (94%) | 375 (94%) | >0.9 |
| Ejection fraction (%) | 21.6 ± 6.9 | 22.2 ± 9.6 | 0.55 |
| Inotropic support | 38 (61%) | 208 (59%) | 0.8 |
| High PVR (>3 Wood units) | 9 (16%) | 72 (21%) | 0.4 |
| BSA ($m^2$) | 1.96 ± 0.25 | 1.93 ± 0.23 | 0.3 |
| Height donor/recipient ratio | 1.00 ± 0.06 | 1.00 ± 0.07 | 0.8 |
| Weight donor/recipient ratio | 1.03 ± 0.26 | 10.1 ± 0.26 | 0.6 |
| Ischemic time >240 min | 0 | 22 (6%) | 0.06 |
| Ischemic time (min) | 161 ± 45 | 161 ± 79 | >0.9 |

*The p value compares patients with donor LVH with donors without donor LVH.
BSA = body surface area; IVS = interventricular septum; LVH = left ventricular hypertrophy; NYHA = New York Heart Association; PVR = pulmonary vascular resistance; PW = posterior wall; WT = wall thickness.

LVH, the vast majority of patients with LVH (11 or 92% vs. 64 or 47%) died from noncardiac causes after 30 days.

**Changes in left ventricular thickness at 1- and 5-years' follow-ups.** Figures 3A and 3B show the comparison of IVS and PW thickness measurements for patients with donor LVH and those without LVH at baseline and at follow-up. Between the groups, significant differences in the donor hearts' wall thicknesses were found only at baseline. At follow-ups (1 and 5 years), wall thickness was similar in the 2 groups. Left ventricular (LV) thickness regression occurred in recipients of allografts with LVH at follow-up (Fig. 4A). Left ventricular wall thickness regression occurred in both IVS (1.28 ± 0.18 cm vs. 1.10 ± 0.13 cm vs. 1.13 ± 0.14 cm, for baseline, 1 year, and 5 years; $p < 0.001$ for change from baseline to 1 and 5 years) and PW (1.27 ± 0.19 cm vs. 1.11 ± 0.11 cm vs. 1.13 ± 0.14 cm; $p < 0.001$ for change from baseline to 1 and 5 years). Rates of post-transplant LVH (38% vs. 32%, $p = 0.5$) were similar in both groups at follow-ups. Patients with or without donor LVH had similar 1-year (3.5% vs. 9.5%, $p = 0.2$) and 5-year (84 ± 5.9% vs. 70 ± 2.7%, $p = 0.07$) survival rates. Both groups had a similar percentage of patients with LVH at 1 and 5 years (Fig. 4B). Sixty-seven percent of the donor LVH group at 1 year and 63% at 5 years no longer met the study's criteria for LVH.

**Table 2 Post-Transplantation Recipient Characteristics**

| | Donors With LVH (n = 62) | Donors Without LVH (n = 365) | p Value* |
|---|---|---|---|
| Hospitalization days | 17.2 ± 22.7 | 16.9 ± 11.4 | 0.4 |
| Tacrolimus/cyclosporine treatment | | | 0.004† |
| Tacrolimus treatment | 25 (40%) | 80 (22%) | |
| Cyclosporine treatment | 37 (60%) | 284 (78%) | |
| Rejection = 3A | 4 (7%) | 44 (13%) | 0.2 |
| Graft CAD | 1 (2%) | 24 (11%) | 0.09 |
| CMV infection | 6 (10%) | 55 (15%) | 0.3 |

*The p value compares patients with donor LVH with donors without donor LVH. †The p value is for both tacrolimus and cyclosporine treatment.
CAD = coronary artery disease; CMV = cytomegalovirus; other abbreviations as in Table 1.

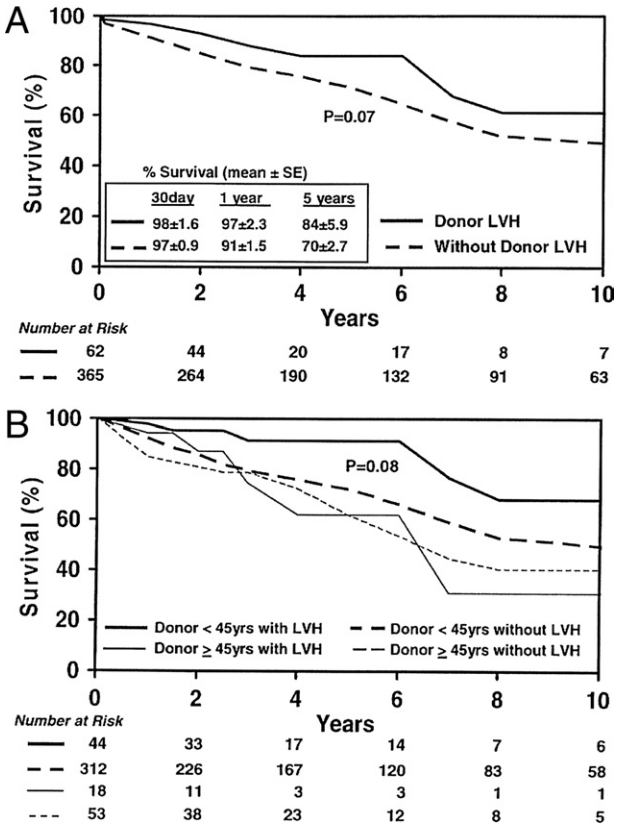


**Figure 1** Survival of Recipients of Donor Hearts With LVH and Without LVH and by Donor Age

**(A)** Recipients of donor hearts with left ventricular hypertrophy (LVH) and those without LVH had similar 30-day, 1-year, and 5-year survival rates. **(B)** Survival analysis of recipients of donor hearts with LVH and without LVH stratified by donor age revealed a trend for better survival among recipients of younger donors' hearts with LVH and without LVH compared with older donors' hearts with LVH and without LVH.

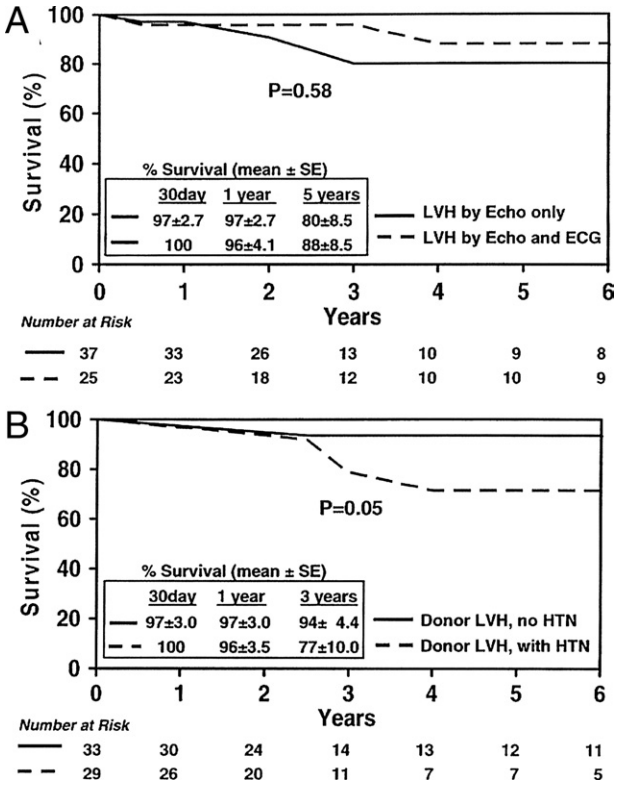


**Figure 2** Survival of Recipients of Donor Hearts With LVH by Echo, Both Echo and ECG, or by Donor History of HTN

**(A)** Survival of recipients of donor hearts with left ventricular hypertrophy (LVH) as determined by echocardiography (echo) alone did not differ from those determined by both echo and electrocardiogram (ECG). **(B)** For approximately the first 2.5 years post-transplant, almost identical survival was observed among recipients of donor LVH with and without donor history of hypertension (HTN); however, those with donor history of HTN have a trend for worse survival thereafter. Summary tables **(insets)** provide survival estimates (±SE) at 30 days, 1 year, and 5 years, respectively.

**Comparison of blood pressures and antihypertensive medications between recipients of allografts with LVH and without LVH.** As presented in Figure 5A, similar systolic and diastolic blood pressures were observed in the 2 groups at 1 year and 62% of all patients had blood pressure <140/90 mm Hg. Antihypertensive post-HTx management treatment is presented in Figure 5B. A similar distribution of treatments using angiotensin-converting enzyme inhibitors (ACEIs), angiotensin receptor blockers (ARBs), and/or calcium channel blockers were found in both groups. Overall, the vast majority (76%) of patients received either single or combination drug management treatments.

**Table 3** Cox Proportional Hazards Survival Model

| Variables | Hazard Ratio | 95% CI | p Value |
|---|---|---|---|
| Older recipient age (>55 yrs) | 1.81 | 1.25–2.62 | 0.02 |
| Race (African American) | 1.86 | 1.15–3.02 | 0.02 |
| BMI (>27 kg/m$^2$) | 1.61 | 1.11–2.34 | 0.03 |
| Donor LVH | 0.49 | 0.26–0.92 | 0.04 |
| UNOS status (2) | 0.66 | 0.46–0.96 | 0.03 |

BMI = body mass index; CI = confidence interval; UNOS = United Network for Organ Sharing; other abbreviations as in Table 1.

## Discussion

This is the first study to evaluate the long-term outcomes of HTx using donors with LVH and to assess the changes in LVWT over time. The main findings of this study are that donor LVH did not have a negative influence on short- or long-term survival; the degree of LVH (mild to moderate) did not affect the survival; both groups showed similar rates of post-transplantation complications; and regression of LVH was obtained over time in recipients with donor LVH.

Donor availability remains the main limiting factor in heart transplantation; as a result, approximately 2,000 transplants are performed annually in the U.S. (1). There is little data regarding HTx of donor hearts with LVH (8,9).

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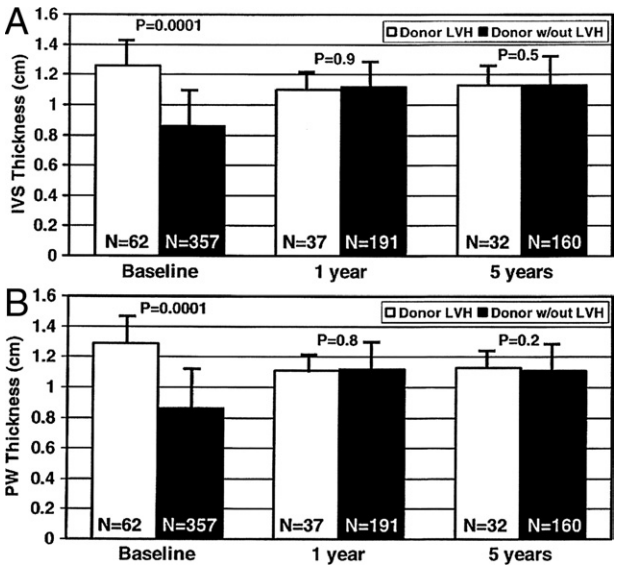


**Figure 3** **Comparison of LV IVS and PW Thicknesses in Recipients of Donor Hearts With and Without LVH**

**(A)** No significant differences of left ventricular (LV) interventricular septum (IVS) wall thickness in recipients of donor hearts with and without left ventricular hypertrophy (LVH) were obtained at 1- and 5-year follow-ups. **(B)** At follow-ups (1- and 5-year), wall thickness was similar in patients with donor LVH and those without LVH. PW = posterior wall.

Recent recommendations by Zaroff et al. (6) discourage the use of donors hearts with more than mild LVH when both echocardiographic (>13 mm) and ECG evidence of LVH is present.

**Survival and post-transplant complications.** Until now only 2 small studies reported the short-term outcomes of HTx recipients with donor LVH (8,9). Marelli et al. (8) evaluated outcomes of 37 patients with donor LVH, but in their study, direct measurements of wall thickness by echocardiography were available for only 6 patients. Lower 1-year survival has been found in those with donors having a history of HTN, ischemic time >180 min, LVH indicated by both ECG and echocardiography, and in recipients with LVH greater than mild or unknown. Aziz et al. (9) reported 30-day outcomes of 9 patients who received allografts with LVH (wall thickness >11 mm).This study demonstrated that the presence of donor LVH increased the incidence of early graft dysfunction (9). In contrast to these 2 reports, our study has a significantly larger number of recipients with donor LVH and importantly had a quantative assessment of LVWT. In addition, the median follow-up was much longer (3.8 years, range 0 to 16.2 years). In contrast to previously published data (8,9), no significant differences in 30-day and 1-year mortality were found between patients who received allografts with LVH compared with those without LVH. Of note, all recipients with donor LVH had ischemic time <240 min. Survival curves extending more than a decade showed no significant differences between recipients with and without donor LVH; moreover, a trend toward better survival for those with LVH was observed. Multivariable Cox regression analysis found that donor LVH was not associated with risk of death and even had some protective effect.

One-third of the donors with LVH had evidence of LVH on both echocardiogram and ECG. Recipients of hearts with LVH determined by both measures did not show higher mortality compared with patients with donor LVH determined by echocardiography alone, which contrasts with the previous report (8). However, donor LVH along with a history of HTN was found to have some negative impact on late post-HTx survival supporting the published data (8). Survival analysis revealed a trend that indicated a possible difference among recipients of younger donors (<45 years) with or without donor LVH and older donors with or without LVH.

Patients who received hearts with mild LVH, moderate-severe LVH, and no LVH had similar survival; however, the number of patients with severe donor LVH was too small to make any statement regarding this particular subset.

**Changes in LVWT, LVH regression, and associated factors.** In this study, we found that LVWT at follow-ups (1 and 5 years) was similar in the 2 groups. Regression of LV thickness occurred in recipients of donors with LVH.

About one-third of all recipients of donors with and without LVH had hypertrophy at follow-ups. Previous

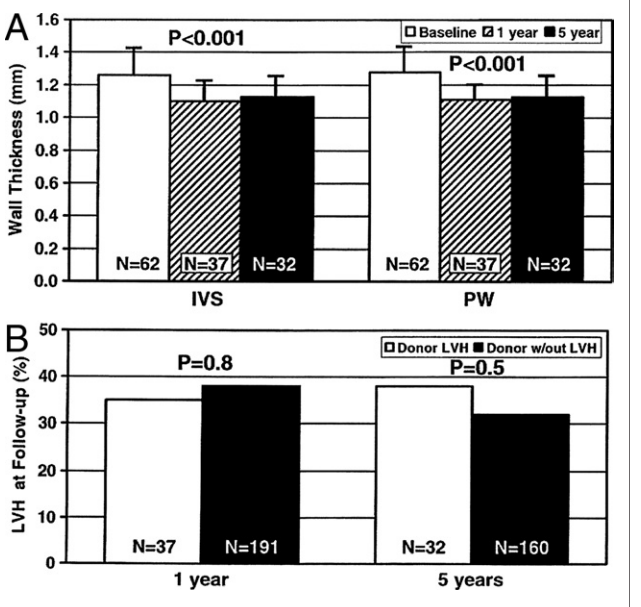


**Figure 4** **LVWT After HTx With Donor LVH, and LVH Prevalence With and Without Donor LVH After HTx**

**(A)** Left ventricular wall thickness (LVWT) regression occurred in both interventricular septum and posterior wall at 1- and 5-year follow-ups. The p value refers to change from baseline to both 1- and 5-year follow-ups. **(B)** Recipients of donor hearts with LVH and without LVH had a similar percentage of patients with LVH at 1- and 5-year follow-ups. Abbreviations as in Figure 3.

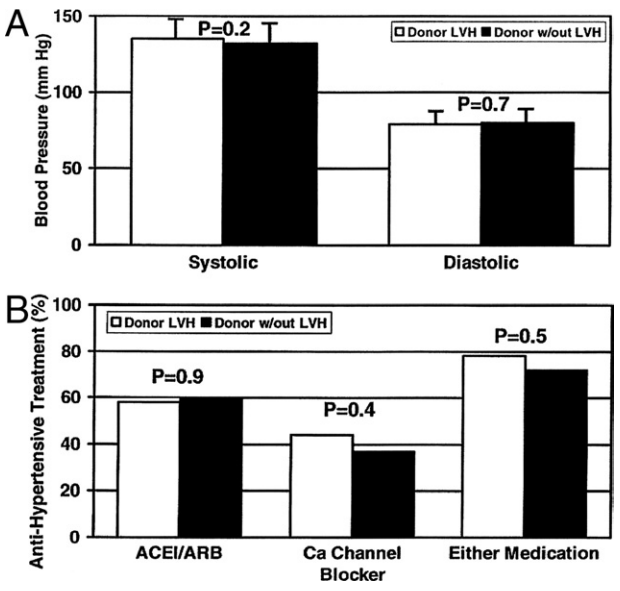


**Figure 5** **BP at 1 Year Post-HTx, and After Treatment in Recipients of Donor Hearts With and Without LVH**

**(A)** Recipients of donor hearts with left ventricular hypertrophy (LVH) and without LVH had similar systolic and diastolic blood pressures at 1-year post-transplantation. **(B)** A similar distribution of treatment with ACEI and/or ARB and Ca channel blockers was found among patients with donor LVH and without LVH. ACEI = angiotensin converting enzyme inhibitor; ARB = angiotensin receptor blocker; BP = blood pressure; Ca = calcium; HTx = heart transplantation.

studies reported various incidence of post-transplantation LVH from very low 13% to surprisingly high 83% at 1 year based on LV mass criteria (13,14). Development of LVH in the post-HTx heart involves multiple factors, including HTN and immune injury (12). Although HTN is common after HTx, occurring in 74% of recipients at 1 year according to the International Society for Heart and Lung Transplantation registry (12), highly variable prevalence of LVH in this population may be related to different cutoff values of LV mass used for diagnosis of LVH (12,13,15).

Two-thirds of our entire cohort received treatment to maintain blood pressures in the normal range (<140/90 mm Hg). As has been demonstrated previously, in the general population with HTN as well in post-HTx patients, these antihypertensive drugs can prevent or even lead to regression of LVH (16–21). Moreover, ACEI and/or ARB therapy has been demonstrated to lead to a regression of LVH regardless of blood pressure control.

Thus, the favorable outcomes of recipients of donor hearts with LVH in our study can be explained by a number of factors: 1) Although donors with LVH were on average older than those without LVH, they were still relatively young; therefore, it is less likely that they had long-standing hypertension. 2) No patients had ischemic time longer than 240 min, the presence of which can affect the outcome in optimal donors, but probably more so in those with LVH. 3) On follow-up, a relatively low proportion of patients had LVH after surgery, which has a potentially negative effect on late survival. 4) The aggressive treatment of HTN and wide use of medications that have been shown to have a beneficial effect on LV remodeling may play an important role in outcomes among recipients with donor LVH.

**Practical implications.** The main issue raised in this study is whether all donors with LVH assessed by echocardiography should be excluded from the potential donor pool. The recent recommendations suggest use of only donors with mild LVH (<1.3 cm). This was based on a small study (8) with short-term follow-up where quantitative assessment of most patients was not available. In our study, we found good short- and long-term outcomes among recipients who received allografts with mild-to-moderate LVH (1.2 to 1.7 cm). Of note, our findings should not be extrapolated to longer ischemic times (>240 min). In addition, the incidence of donor diabetes mellitus was very low and not always available; therefore, our results mostly apply to nondiabetic donors with LVH. Although the overall outcomes of patients with donor LVH are similar to those without LVH, recipients of younger donor hearts with LVH had the best survival rates, leading us to think that most of them had physiologic LVH. A history of donor HTN in those recipients with donor LVH was found to have an unfavorable impact on late survival, suggesting that those patients had more advanced changes in myocardial structure and diastolic function.

This last finding brings up the next important question: How can we identify donors in whom LVH is accompanied by significant diastolic dysfunction and is less likely to regress, which can affect the outcome detrimentally? Among those with LVH, it is known that healthy, young athletes may have physiologic hypertrophy with normal systolic and diastolic LV function (22). These hearts are different from hearts of older donors with severe LVH, with long-standing HTN with impaired diastolic and even systolic function. Recently, echocardiographic techniques such as tissue Doppler imaging, strain, and strain rate have been introduced for comprehensive LV function assessment. Use of these new techniques should be evaluated in HTx in the future and may be extremely useful for transplant decision making in donors with moderate-to-severe LVH.

**Study limitations.** The major limitation of our study is its retrospective nature. However, this study includes the largest number of patients who have received allografts with LVH. Moreover, this is the first study that used precise measurements of LV thickness. Another limitation is the lack of calculation of LV mass. The formula for LV mass calculation requires cubing of several primary measurements (11). In a retrospective study where not all measurements are made by a single operator, even small errors in these measurements can be magnified; therefore, only primary measurements were included.

## Conclusions

Recipients of transplanted donor hearts with LVH had similar short- or long-term survivals to those who received donor hearts without LVH. These results suggest that donor hearts with mild and moderate LVH can be safely used for heart transplantation and may increase the number of hearts available for transplantation. The lack of progression or even regression of LVH can be achieved in recipients of donor hearts with LVH by optimal blood pressure control and treatment using drugs that have shown a beneficial effect on LV remodeling.

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